



Sea World

HAND DELIVERED

September 12, 1990



Dr. Nancy Foster
Director, Office of Protected
Resources and Habitat Programs
National Marine Fisheries Service
1335 East-West Highway, Room 8268
Silver Spring, Maryland 20910

RE: Marine Mammal Collection/Inventory Reports

Dear Dr. Foster:

Two copies of the attached serve as an update to our
inventory reports.

Sincerely,

Barbara D. Heffernan
Director, National Affairs
1776 I Street, N.W. Suite 200
Washington, D.C. 20006

cc: Mr. Edward Asper
Mr. Brad Andrews

Attachment: Marine Mammal Collection/Inventory Reports
SWC-Pc-8729 NMFS Inventory/Mortality Report
SWC-Pc-8729 Sea World Gross Necropsy Report

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Corporation
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P2P

SN:	ASN:	LEX:
SP:	ANREP:	FNUM:

MARINE MAMMAL COLLECTION/INVENTORY REPORT

COMMON NAME: False Killer Whale

[illegible]

SEA WORLD
GROSS NECROPSY REPORT

FACILITY: Sea World of California PROSECTOR: Jim McBain, D.V.M.
GENUS/SPECIES: Pseudorca crassidens
ID NUMBER: SWC-PC-8729/ SW 90044 AGE: adult SEX: female
DATE OF DEATH: 6/16/90 DATE OF NECROPSY: 6/16/90
WEIGHT: 444 kg TOTAL LENGTH: 378 cm

HISTORY: This animal was demonstrating diminished activity and irregular appetite. A blood sample was taken on 5/30/90. An elevation of the white count was noted. Due to indications of bacterial infection, the animal was started on antibiotic therapy. After five days a satisfactory response had not been demonstrated, so a change in therapy was instituted. Total white count began to drop, but the differential count did not improve, and a worrisome elevation of liver enzymes and bilirubin along with a decrease of serum proteins was evident. Further diagnostic tests revealed a need to maintain antimicrobial therapy in spite of a falling white count. Appetite and activity remained at a low level for a week. On 6/11/90 no appetite was apparent. Medication and caloric support were given via stomach tube for the next week. Continued blood tests showed further deterioration of the animal's condition. The medication regimen was constantly adjusted to accommodate the negative changes in the clinical picture. This was all to no avail. The liver enzymes continued to rise, the bilirubin levels continued to rise, and serum protein levels continued to drop. On 6/16/90 the animal developed a list and died at 5:30 p.m.

Code, in parentheses, for samples taken:

C = culture; V - virology; M = metals;
P = pesticides; E = electron microscope samples

GENERAL EXTERNAL APPEARANCE: (oral cavity, external nares, skin, eyes)
Some peeling is present on the animal's dorsal aspect between the dorsal fin and the blowhole. There is a skin blister approximately 6 cm in diameter left of the dorsal midline one-half the distance between the dorsal fin and the blowhole. The vesicle involves only the outer layers of epidermis and does not extend to the dermis. There is a healed tooth extraction at the position of the fourth lower right tooth.

SUBDERMAL CONDITION: (blubber, muscles, lymph nodes)

The blubber layer on the ventral midline is 4 cm thick. Over the right thorax it is 2.8 cm thick. There is edema of the tissue between the blubber and muscle on the dorsal aspect of the animal between the dorsal fin and the blowhole.

CENTRAL NERVOUS SYSTEM: (brain, pituitary, spinal cord)THORACIC CAVITY: (pleura)

There is approximately 250 ml of clear yellow fluid in each hemithorax.

LOWER RESPIRATORY SYSTEM: (trachea, bronchi, lungs, lymph nodes)

The trachea and main stem bronchi are full of thick white foam which originates from the lung parenchyma. The lung color is normal and no firm areas are palpable. White foam exudes from the bronchi on cut section. The right lobe weighs 2.3 kg, the left lobe weighs 2.7 kg. The bronchial lymph nodes are edematous and exude watery clear fluid on cut section.

ABDOMINAL CAVITY: (lymph nodes)

The abdomen contains 10-12 liters of clear yellow fluid. The abdominal lymph nodes are large and edematous. The omentum is hemorrhagic and edematous.

DIGESTIVE SYSTEM: (esophagus, stomach, intestine, rectum, cecum, lymph nodes)

The esophagus has two small, 1-2 cm long, healing mucosal lacerations. There is very little inflammation and no bleeding associated with these lesions. The gastric mucosa looks normal. The muscularis is very hemorrhagic and edematous. The duodenum, especially proximally, is distended and full of bile-tinged mucus. The mucosa appears normal. The jejunum and ileum have a slightly firm feeling and very small diameter compared to the duodenum. The wall is very thickened (6 mm), and the lumen is almost nonexistent. The mucosa looks normal, and the lumen is almost empty. The total diameter of intact jejunum is about 1.5 cm with occasional bulges up to 2.0 cm. The ileum appears grossly the same as the jejunum. The color appears normal. The mesenteric lymph nodes are enlarged and drop watery fluid from the cut surface.

LIVER: (biliary system)

The liver weighs 7.820 kg. It is slightly swollen, with rounded edges. The color is a uniform light chocolate brown.

SPLEEN: The spleen weighs 70 gm and looks grossly normal.REPRODUCTIVE SYSTEM: (testicles, ovaries)

The uterus and ovaries look normal with a single luteal scar on the right ovary.

URINARY SYSTEM: (kidneys, ureter, bladder, urethra)

The right kidney weighs 1.93 kg, and the left weighs 2.04 kg. The kidneys look grossly normal. Dark greenish urine was taken from the bladder.

SPECIAL TESTS: Histopathology of heart, lung, liver, intestine, spleen, lymph node, stomach.

GROSS SUMMARY:

- 1) Pulmonary edema;
- 2) Ascites;
- 3) Esophageal lacerations;
- 4) Gastric edema;
- 5) Hypertrophy of muscularis of duodenum and ileum;
- 6) Hepatic fatty degeneration.

TENTATIVE DIAGNOSIS: Death is due to hypoxia associated with pulmonary congestion.

CONCLUSIONS: (after histology & clinical pathology review)

There is histologic evidence of severe lack of lymphoid activity, which has been confirmed clinically by a chronic lymphopenia. A significant decrease in serum gamma globulin was seen clinically, which can be attributed to a decrease in B-lymphocyte activity. The hypoproteinemia that resulted from the B-lymphocyte suppression is responsible for the fatal pulmonary edema. Clinical laboratory and histological evidence indicate that failing liver function was also contributing to the deterioration of the animal.

DATE:

8/20/90

SIGNED:

Tim R. Campbell